

Diseases of Sebaceous and Sweat glands

- Acne vulgaris
- Rosacea
- Rosacea-like disorders:
 - 1. Solid facial edema
 - 2. perioral - periorcular dermatitis
 - 3. Papulopustular Eruption
 - 4. Rosaciform dermatitis
 - 5. Steroid Rosacea
 - 6 - pityriasis folliculorum
 - 7 - Haber's Syndrome
- Lupus Miliaris (acne agminata)
- Fordyce spots
- Miliaria (Sweat Rash)
- Bromhidrosis
- chromhidrosis
- Hyperhidrosis
- Anhidrosis
- Granulosis Rubra Nasi
- Fox - Fordyce disease

☀️ Rosacea ☀️

- D.F: Chronic vascular inflammatory Disorder - limited to Center of face.

ch.ch By: 1- persistent Erythema
2- Telangeectasia
3- papules - pustules

- Epidemiology:

- 30-50 yr - female more -
- Incidence Highest in skin types I-II
- Lowest in darkly pigmented skin

- Clinically:

1. affect mainly Center of face →
cheeks - Chin - forehead - Nose
(Blush area of face)

2- Rarely may affect :- arms - legs
Chest - Scalp - Scrotum

(Red scrotum Syndrome)

3- Skin → usually Oily ☐

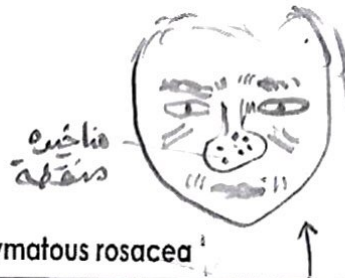
- 1ry - 2ry Features of rosacea:

1ry

- 1- Flushing (Transient Erythema)
- 2- Non-Transient Erythema
- 3- papules - pustules
- 4- Telangeectasia

2ry

- 1- Burning - stinging (malar skin)
- 2- Dry appearance (central facial skin)
- 3- soft or solid facial edema
- 4- Ocular manifestation
- 5- peripheral location
- 6- Phymatous change



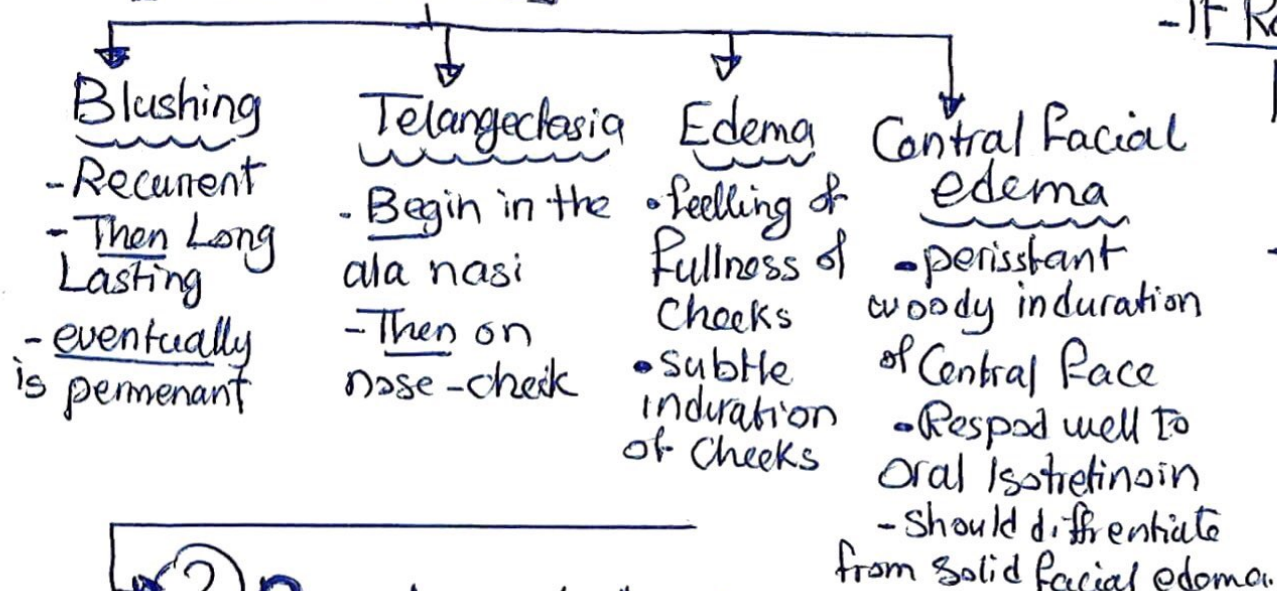
Types of phymatous rosacea

Rhinophyma (Fig. 52)	<ul style="list-style-type: none"> • Apparent initially as dilated patulous follicles at the distal end of the nose. • When marked, can lead to debilitating nasal deformity.
Gnathophyma	<ul style="list-style-type: none"> • Rare occurrence, with central chin typically involved. • May give rise to asymmetrical swelling.
Otiophyma	<ul style="list-style-type: none"> • Usually affects the lower half of the helices & lobes of the ears.
Mentophyma	<ul style="list-style-type: none"> • Cushion-like, firm swelling of the central forehead.
Blepharophyma	<ul style="list-style-type: none"> • Swelling of the eyelids. • Usually seen as a component of edematous rosacea but may accompany severe papulopustular or ocular rosacea.

- Rosacea subtypes:

① Erythematotelangiectatic:

[vascular rosacea]



- it appears as: Red deep irregular Lobulated thickening of skin of the Nose with Follicular dilatation

- it Represent:

Hyperplasia of Sebaceous gland and connective tissue

- phymatous process may develop: in extranasal sites as: Chin - forehead - cheeks - ears

② Papulopustular:

[Inflammatory Rosacea] PPR

- varies from: Small papules - pustules to occasional deep peristent Nodules
- No comedones - No Scarring
- Papules are Asymptomatic

③ phymatous rosacea:

- Seen in Long lasting Cases in men

④ Ocular rosacea

- Can occur without skin lesion. (Difficult to Diagnose)

- Mild: Scaling of eyelid margin
- More Active: Blepharitis
 - Conjunctival injections
 - Cysts from meibomian gland (chalazion)
- Severe: Keratitis • Uveitis
- Corneal neovascularization

- Rosacea Variants:

a) Granulomatous rosacea

- more persistent
- discrete
- red to red-brown facial papules and nodules

b) Pyoderma faciale:

(Rosacea fulminans)

- affect: post-adolescent women
- 20-40 yr
- with NO previous history of Acne
- ch.ch By: sudden erupted purulent nodulocystic lesions
- specialty in: Face, NO systemic
- NO comedone
- The Back, chest → Free.
- History of: Flushing
- treatment: Tetracycline (1g/day)
or Isotretinoin (1mg/kg)

with intralesional Steroids or liquid Nitrogen to the Cyst

- Systemic Steroid Dose of 40-60 mg daily
 tapered → over 3-4 weeks.

(This is one of the few times that oral steroid should be used in ~~the~~ of rosacea)

- Histopathology:

- ch.ch By: 1- Vascular Dilation
2- Perivascular non-specific inflammatory infiltrate
3- Solar elastosis
- in 10% of papular lesions:
"Tubercloid" picture found $\left\{ \begin{array}{l} \text{epithelioid cells} \\ \text{giant cells} \end{array} \right.$
- This leads to Diagnosis of: Rosacea-like Tubercloid
- Presence of granulomatous formation in rosacea is explained as: Foreign Body reaction against Keratinized cells or disintegrated hair structures or against elastic material.
- The granuloma may represent: Delayed Hypersensitivity Reaction to mite Demodex folliculorum

[3]

- Dermoscope:

- 1- Dilated Vessels
- 2- Prominent telangiectasia
- 3- Large polygonal vascular Net.

Pathogenic hypotheses of rosacea

Aberrant innate immune system	<ul style="list-style-type: none"> The innate immune response protects against microbial infection without requiring specific recognition of the pathogenic stimulus. Activation of innate immunity leads to release of cytokines & antimicrobial molecules such as the peptide cathelicidin. In rosacea, there is upregulation of cathelicidin and its processing serine protease, suggesting dysfunction of the innate immune system.
Ultraviolet radiation	<p>UVR exposure has been shown to:</p> <ul style="list-style-type: none"> Induce angiogenesis. Increase production of reactive oxygen species, which then leads to upregulation of matrix metalloproteinases (MMPs result in damage to blood vessels and the dermal matrix).
Vascular changes	<ul style="list-style-type: none"> An increase in blood flow is seen within affected skin in rosacea. Rosacea patients flush more readily in response to heat as compared with controls. An elevated expression of VEGF & lymphatic endothelial markers is observed, suggesting stimulation of blood vascular & lymphatic endothelial cells.
Epidermal barrier dysfunction	<ul style="list-style-type: none"> Increased transepidermal water loss. Lowered threshold for skin irritancy. Seen in both erythematotelangiectatic and papulopustular rosacea.
Microbes	<ul style="list-style-type: none"> Demodex mites (folliculorum & brevis), which are commensals of normal skin, are found in greater numbers in rosacea patients. Demodex infestation is associated with an intense perifollicular infiltrate of predominantly CD4+ helper T cells. Other microorganisms which could be involved in the pathogenesis of rosacea: Staphylococcus epidermidis, Helicobacter pylori, Bacillus oleronius & Chlamydia pneumoniae.

- Proposed role for micro-organisms in rosacea.

Step I:-

- unaffected rosacea-prone skin has ↑↑ expression of microbe sensing machinery and normal flora

Step II:-

- Commensal agents may act as trigger factors for rosacea inflammation through activation of pattern recognition receptors (PRR) or altered tolerance.

Step III:-

- physiological and inflammatory changes in rosacea skin → Create a micro-environment → suitable for altered growth or metabolism of microbes

Step IV:-

- alteration in commensal flora or
- introduction of non-commensal agents → activation of inflammatory pathway.

- Step V: Immune Response that result in clearance of exacerbating agent → impair temporary relief

-Exacerbating Factors:

- 1- Sun exposure
- 2- Alcohol
- 3- Tea-Coffee: it is the temperature of the coffee-tea Not The Caffeine
- 4- Psychic Factors

-Treatment:

• General •

- 1- avoid extreme Hot - Cold
 - excessive sunlight
 - Spices
 - Hot liquids Alcohol

• Systemic •

1- Tetracyclines:

- 250 mg / 3 times / daily
- 3-4 weeks
- 2nd generation Tetracyclines are effective
 - Minocycline
 - Doxycycline

2- Erythromycin: (200-250-500 mg)

- 30-50 mg/kg / day
- 2nd generation Macrolides → azithromycin
→ Clarithromycin

3- Metronidazole: Flagyl

200 mg / once or twice daily For 4-6 weeks

4- Isotretinoin: (Accutane) 10-40 mg / daily

• Topically •

- 1- Metronidazole → 0.75% - 1% Cream - gel - Lotion
- 2- Sodium Sulfacetamide → 10% Cream - Foam - Lotion - wash
- 3- Azelaic acid → 15% gel
- 4- Brimonidine → 0.33% gel (Mirvaso)
 - For: persistent nontransient Facial Erythema of rosacea in Adult 18 yr or Older.
 - Brimonidine tartrate → Highly selective α -2- adrenergic receptor agonist with Vasoconstrictive activity → In H of Glaucoma
- 5- Ivermectin → 1% Cream (Soolantra)
for H of inflammatory lesions of rosacea.

6 - Pimecrolimus → 1% cream
twice-Daily

7 - Tacrolimus → twice daily
with 100 minocycline/twice

8 - Benzoyl peroxide /
Clindamycin :-

• Plastic Surgery For Rhinophyma •

- Electrodesiccation OR
Tunable dye laser for
telangiectasia

• Laser-Light Therapies •

① Vascular Laser Therapy :-

- The standard pulsed Dye laser
- The Long-pulsed dye laser
- The diode-pumped frequency
doubled Laser

② Intense pulsed-Light therapy

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③ CO₂ laser : For phymatous Rosacea.

④ Nd-YAG laser + PDL :- Recently proved to be safe
and effective in the treatment of Erythematotelangiectatic rosacea.
and ~~the~~ in the concentration of substance P → implicated in
pathogenesis of rosacea.

• Facial skin care in rosacea patient •

- ① wash with lukewarm water
- ② use Soap-free cleansers
- ③ Cleanser are pH balanced and applied gently e finger tips
- ④ Use Sunscreen with Both UVA + UVB protection and SPF > 15
- ⑤ Sun Blocking Creams :- Containing the physical Barrier:
Titanium dioxide or/and Zinc oxide
- ⑥ use Cosmetics and sunscreens that contain protective Silicones
- ⑦ water-soluble facial powder contain :- inert green pigment
→ helps to neutralize the perception of Erythema
- ⑧ Moisturizers :- Containing humectants e.g: glycerin.
Occlusives : e.g: petrolatum → help Repair Epidermal Barrier
- ⑨ Avoid → astringents - toners - abrasive exfoliators
- ⑩ Avoid → Cosmetics that contain alcohol, menthols,
Camphor, witch hazel, fragrance, peppermint
- ⑪ Avoid → Waterproof cosmetics and heavy foundations
That's difficult to Remove about irritations

Treatment of rosacea

Type of rosacea	Topical	Systemic	Other
Papulopustular	<ul style="list-style-type: none"> Wide-spectrum antibiotics. Tretinoin. Metronidazole. 	<ul style="list-style-type: none"> Wide-spectrum antibiotics. Isotretinoin. Metronidazole. 	<ul style="list-style-type: none"> Pimecrolimus (1% cream) twice daily.
Erythematotelangiectatic	<ul style="list-style-type: none"> Topical agents, e.g. azelaic acid, metronidazole & topical oxymetazoline (selective α_1-agonist). 	<ul style="list-style-type: none"> Propranolol. Clonidine. 	<ul style="list-style-type: none"> Laser.
Ocular	<ul style="list-style-type: none"> Wide-spectrum antibiotics. Cyclosporine 0.5% ophthalmic emulsion. Metronidazole gel. 	<ul style="list-style-type: none"> Wide-spectrum antibiotics. 	<ul style="list-style-type: none"> Liquid paraffin eye ointment. Eyelid hygiene & artificial tears.
Rhinophyma	-	<ul style="list-style-type: none"> Isotretinoin May reduce nasal volume and halt the progression of rhinophyma. 	<ul style="list-style-type: none"> Surgical / laser remodeling.
Pyoderma faciale	-	<ul style="list-style-type: none"> Oral steroids Isotretinoin. 	-

- Emerging therapies in Rosacea:

I Systemic therapy:

↳ @ Sub-antimicrobial - dose Doxycycline :-
(20 mg / twice/day or once daily 40 mg)
act Through:

1- Down Regulation of Cytokines with Reduction of Neutrophils infiltration and deactivation of Downstream inflammatory pathway

2- inhibition of nitric oxide & Reduction of Vasodilatation and cessation of Capillary wall degradation → Reduce leakage

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3- Reduction of level of reactive Oxygen Species → slow destruction of Connective Tissue

4- inhibition of matrix Metalloproteinase

↳ (b) B-Blockers: act on Flushing:

1- Blocking the B-adrenergic Receptors on smooth muscles of cut. Blood vessels.
→ vasoconstriction

2- Reduce anxiety, tachycardia → which can exaggerate flushing reactions.
Carvedilol → newer nonselective B-Blocker & marked antioxidant and anti-inflammatory action.

↳ (c) Oral Ivermectin:

Single Dose - oral Ivermectin → microfilaricide → used in immunocompromised pts & rosacea like demodicidosis.

II) Topical therapy:

- all are Targeted to control of Demodex folliculorum and D. brevis mites.
- Permethrin 5% - Crotamiton 10%
Ivermectin 1%.

- Rosacea-like disorders:

[1] Solid facial edema (morbihan's disease)

[2] perioral - perioral dermatitis :-

- in young females
- lesion: Persistent Erythematous Eruption of tiny papules and pustules → around the mouth, nose, eyes.

- a clear zone → seen around the vermilion border of lips

- etiology:

- Due to prolonged therapy of fluorinated Steroids - Sunlight - Oral CPs
- fluorinated tooth paste
- heavy users of cleanser, moisturizers.

- Treatment:

1. Stop → The topical corticosteroid Therapy
2. Systemic antibiotics: tetracycline

Doxycycline - Minocycline - Erythromycin
Azithromycin → 4-6 weeks

3. Topical Metronidazole or Azelic Acid

4. pimecrolimus cream 1% → twice daily

[3] papulopustular Eruptions :-

- Due to → epidermal growth factor Receptor EGFR inhibitors

[4] Rosaceiform dermatitis :-

- facial Erythema - small papules
- numerous pustules
- Caused By: application of topical Calcineurin inhibitors on face.

[5] Steroid Rosacea:

- Fluorinated and potent corticosteroids cause problems (topical or inhaled) → induce and exacerbate Rosacea.
- presence of → Rosacea-like lesion on the upper lip and around ala nasi

↓
Clue that corticosteroid involved.

[6] Pityriasis Folliculorum :-

- Roughened - whitish - scaling skin surface
- superimposed on a background of faint Erythema + scattered fine papules and pustules
- young - middle-age women
- Skin scraping → reveals presence of Demodex mites

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- application of → Topical sulfur preparation OR permethrin 5% at night for 2 weeks → till

[7] Haber's Syndrome:

- rare familiar Granulomatosis
- Ch. ch by: Early Onset facial Erythema - Verrucous papules truncal Keratotic lesions and pitted atrophic scars.

• Lupus Milian's LMDF Disseminatus Faciei • (Acne agminata)

• Lesion:

- Discrete - Reddish papules
- Site: Face mainly on Eyelids cheeks & upper lip.
- Erythema - telangiectasia → The ch. ch of Rosease is Absent
- involute spontaneously in a year → leaving pitted small scars

[9]

- In 2000 → The name changed from LMDF to FIGURE:
facial idiopathic Granulomatous & Regressive Evolution

• Histopathology:

- Large tubercles → composed of:
 - epithelioid cells
 - giant cells → large areas of caseation necrosis in the Center
- at the periphery: inflammatory infiltrate

• Histogenesis:

- No evidence to support tuberculous etiology
- Doesn't Respond to anti-Tuberculous Drugs

• Treatment:

- Long term therapy is • Tetracycline • minocycline OR • Isotretinoin -

• Fordyce Spots •

- D.F: heterotropic Sebaceous glands that can occur:

- ↳ Around the vermilion Border of the lips or within the oral mucosa; Asymptomatic - multiple Symmetrical - barely elevated - Yellow papules
- ↳ ventral surface penile shaft → inflamed
- ↳ Areolar area of the Breast

• treatment:

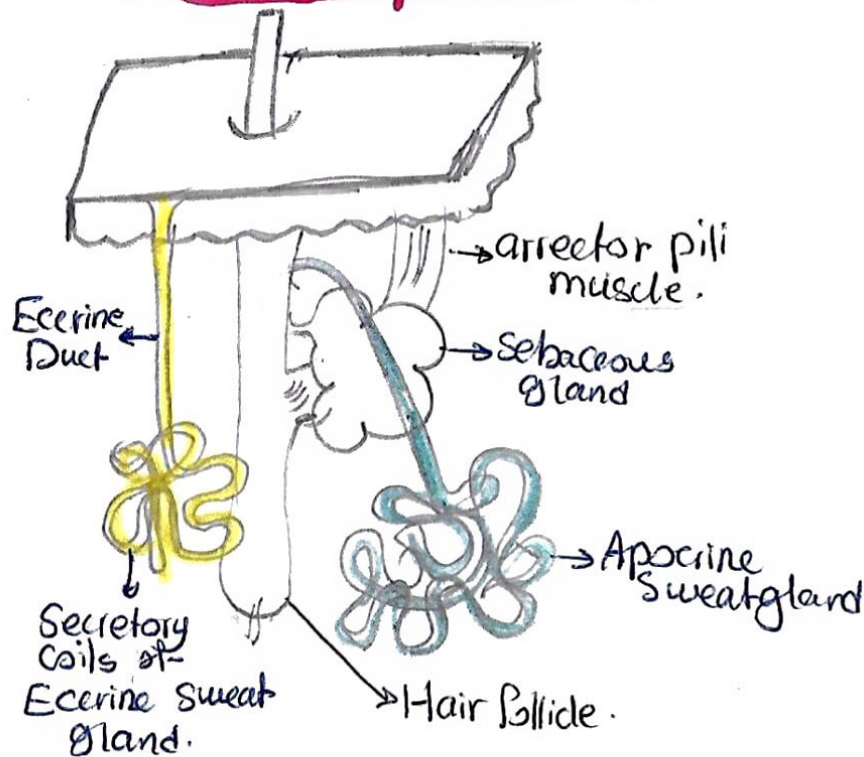
- ↳ Oral Isotretinoin → For extensive lesions
- ↳ Carbon Dioxide ablative laser

Sweat gland

- 2 main types: + 3rd type



Anatomy - physiology:



	Eccrine sweat glands	Apocrine sweat glands	Apoeccrine sweat glands
Localization	<ul style="list-style-type: none"> Entire body skin (except the external auditory canals, vermillion lips, clitoris & labia minora). Highest density on palms & soles. 	<ul style="list-style-type: none"> Axillae, anogenital, periumbilical, nipples. 	<ul style="list-style-type: none"> Axillae.
Embryology	<ul style="list-style-type: none"> From downgrowth of epidermis at 3rd month of IUL independent of pilosebaceous follicle. No new glands develop after birth. 	<ul style="list-style-type: none"> As a part of pilosebaceous follicle in 4th of 5th month of IUL 	<ul style="list-style-type: none"> Not present at birth.
Morphology	<ul style="list-style-type: none"> Long, thin duct opens to skin surface. Secretory coil with narrow lumen. 	<ul style="list-style-type: none"> Short, thick duct opens into upper part of follicular canal. Secretory coil with wide lumen. 	<ul style="list-style-type: none"> Duct comparable to eccrine gland duct. Secretory coil with dilated & non-dilated tubular segments.
Cell types in secretory coil	<ul style="list-style-type: none"> Large secretory clear cells, dark cells, and myoepithelial cells. 	<ul style="list-style-type: none"> Epithelial & myoepithelial cells. 	<ul style="list-style-type: none"> Eccrine & apocrine morphologic features.
Secretion	<ul style="list-style-type: none"> No cellular breakdown, i.e. merocrine glands where the cells are not destroyed during secretion. 	<ul style="list-style-type: none"> Pinching off parts of apical cytoplasm → oily odorless fluid (bacterial decomposition → odors). 	
Main innervation/ neurotransmitter	<ul style="list-style-type: none"> Sympathetic fibers/acetylcholine. 		
Development	<ul style="list-style-type: none"> Present at birth. No relationship to pilosebaceous follicle. 	<ul style="list-style-type: none"> Present at birth. Associated with terminal hair follicle. 	<ul style="list-style-type: none"> Develop during puberty from eccrine-like precursor cells. No relationship to pilosebaceous follicle.
Function	<ul style="list-style-type: none"> Thermoregulation/role in hyperhidrosis and hypohidrosis. 	<ul style="list-style-type: none"> Unclear/some role in olfactory communication. Role in follicular apocrine Fox-Fordyce disease 	<ul style="list-style-type: none"> Thermoregulation/role in axillary hyperhidrosis & non-follicular apoeccrine Fox-Fordyce disease.
Composition of the secretions	Odorless clear fluid: <ul style="list-style-type: none"> pH 4 – 6.8 (hypotonic). H₂O, Na, Cl, K, urea, lactate & glucose. 	Sterile, odorless and viscous: <ul style="list-style-type: none"> pH 5.0 – 6.5. Precursors of odoriferous substances (cholesterol, triglycerides, fatty acids, cholesterol esters, squalene). It also contains androgens, carbohydrates, ammonia & ferric iron. 	<ul style="list-style-type: none"> Unknown.

- Humans have up to 4 million sweat glands over all body.
 3 millions are Eccrine sweat glands
 - Ratio 1:1 = apocrine to Eccrine →
 in axilla 1:10 in elsewhere

❖ Miliaria (Sweat Rash) ❖

1. D.F:

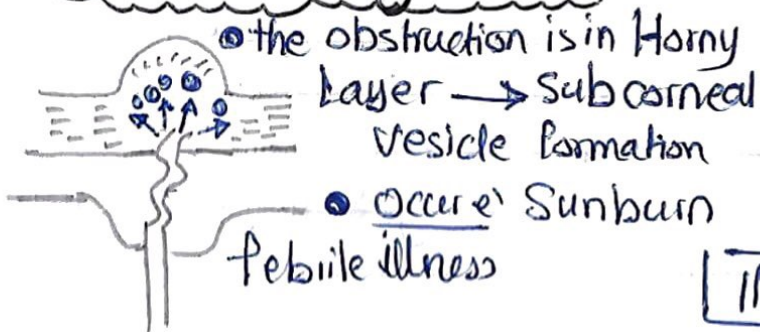
- Common condition after prolonged exposure to Hot-humid environment: tropics. Occupation
- most common in: neonates → (whose eccrine sweat ducts Not fully developed)

-2. Pathogenesis:

- excessive sweating
- under occlusive clothing lead to → maceration of the Stratum Corneum.
- Sufficient to Cause Blockage of eccrine Duct
- Followed By → Rupture of The Duct and Sweating below the level of obstruction

3. Clinically: according to level of obstruction

① Miliaria Crystallina



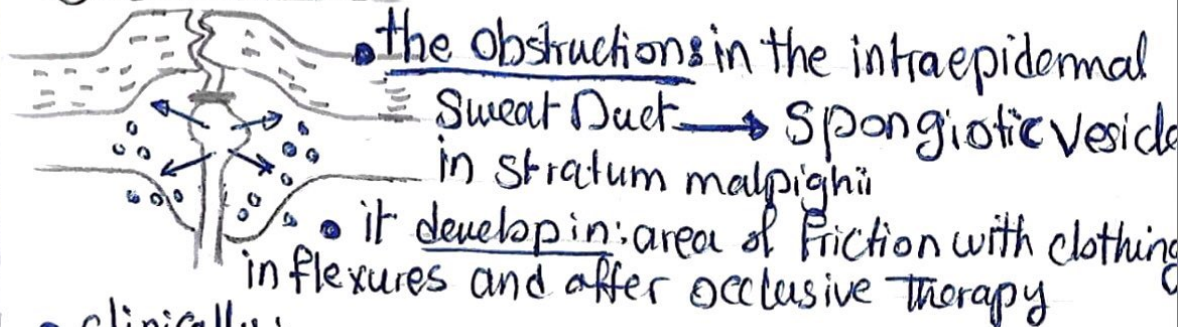
- associated :- profuse sweating, in newborn & excessive clothing and heavy sweating

Clinically:

Asymptomatic - Small - clear - non inflammatory Thin walled Vesicles

- mainly on: Trunk
- Vesicles → Rupture → superficial branny desquamation

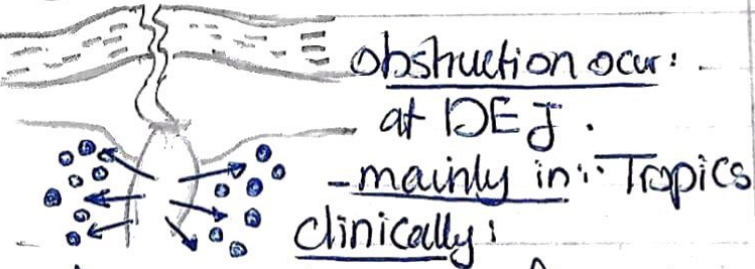
② M. rubra:



Clinically:

- Small - papulovesicles Surrounded by Erythema with pricking sensation
- may be pustular (pustulosa) → in chronic extensive cases
- its Sterile bacteriologically

③ Miliaria Profunda:



Asymptomatic • pale • firm
Papules mainly on Trunk

Lesions:

Asymptomatic because Sweat Retention vesicle that forms → lies in the upper dermis.
Below the level of the Itch Receptors

-Complications:

- ① 2ry bacterial infection → miliaria pustulosa
- ② Disturbance of heat regulation → when Large numbers of glands are occluded and non-functional → these individuals at Risk for significant thermoregulatory Problems and Compensatory Hyperhidrosis of the Face.

Type	Location of obstruction	Cutaneous lesions	Patient	Common locations
Miliaria crystallina	Stratum corneum.	Non-pruritic, clear, fragile, 1 mm vesicles.	Neonates <2 weeks of age. Children & adults in hot climates.	Face & trunk.
Miliaria rubra	Mid-epidermis.	Pruritic, erythematous, 1-3 mm papules; may have pustules.	Neonates 1-3 weeks of age. Children & adults in hot climates.	Neck & upper trunk.
Miliaria profunda	Dermal-epidermal junction.	Non-pruritic, white, 1-3 mm papules.	Adults in hot climates; often with multiple bouts of miliaria rubra.	Trunk & proximal extremities.

③ Axillary and inguinal lymphadenopathy → seen

④ "Tropical anhidrotic asthenia" → applied to These pts

-treatment:

- ① Avoid excessive Sweating. Stay in AC.
- ② wearing Light clothing
- ③ Gradually → The obstructed sweat Ducts Shed their Occluding Keratinous plugs → normal sweating
- ④ Anhydrous Lanolin → prevent Ductal Blockage, allowing sweat to flow to the skin surface.
- ⑤ Occlusive ointments → avoided
- ⑥ Calamine Lotion → for cooling symptomatic relief after miliaria develops.

Diseases of Sweat Glands

1-specific diseases of Sweat gland:

- Miliaria
- Altered composition of eccrine sweat
- Cytologic inclusions and other morphologic changes of Diagnostic importance in sweat gland.

2-Hyperhidrosis

3-Hypohidrosis + Anhidrosis.

4-Sweat as a predisposing influence on skin Disease

5-Apocrine sweat Gland Disease

❖:- Altered Composition of Eccrine :-❖

1] Altered electrolyte Content :- systemic disorders that affect the levels of electrolytes in sweat.

→ ↑↑ level of electrolytes in sweat :-

- * Cystic Fibrosis : ↑ CL ($>60 \text{ mEq/L}$)
- * Endocrine disorders : - Adrenal insufficiency
→ Myxedema
- * Malnutrition

→ ↓↓ level of Electrolytes :-

- * Endocrine disorders :-
→ Aldosteronism
→ Cushing's Syndrome
→ Thyrotoxicosis
- * Hypoproteinemic edema

2] Bromhidrosis :-

→ Apocrine bromhidrosis

- Axillary : penetrating odor
D.T → Bacterial decomposition of apocrine sweat mainly :-
Axilla > e' puberty
- The apocrine sweat : originally odorless, apart of excreted substances as garlic.
- Androgens : of apocrine sweat → gives rise to odoriferous substances (pheromones)

→ Eccrine bromhidrosis

- | <u>Keratogenic</u> | <u>Metabolic</u> | <u>Exogenous</u> |
|---|--|--|
| <ul style="list-style-type: none"> • 2ry to maceration of st. Corneum with bacterial degradation of Keratin - plantar or Intertriginous | <ul style="list-style-type: none"> • phenylketonuria (musty-Mousy odor) • Maple syrup Urine disease (Sweat odor) | <ul style="list-style-type: none"> • Foods :
- garlic - Curry asparagus • Drugs :-
penicillin - bromides • Chemicals :
dimethyl sulfoxide |

● Treatment of Bromhidrosis :-

- 1- frequent washing of axillary region
- 2- ↓ Foods as garlic from diet
- 3- Local: Antibacterial substances
aluminium salts
- 4- frequency - doubled, Q-switched
Nd:YAG laser → effective
- 5- Botulinum toxin A → future therapy

[3] Chromhidrosis :-

- Apocrine in origin
- Found in: genital - axillary - areolar
facial skin
- chromhidrosis Reported only: on
The face, axillae, breast areola.
- lipofuscin pigment → responsible
for the colored sweat.

↓
This pigment is produced by Apocrine

- The various Oxidative states →
Responsible for the ch.ch → yellow
green - Blue - Black secretion
observed in apocrine chromhidrosis

- Eccrine Chromhidrosis: is Rare, occur with:
Digestion of certain dyes or Drugs: - Red sweat in
Ptn receiving Clofazimine - Rifampicin.

- Pseudo-Chromhidrosis: when clear eccrine sweat
become colored on the surface of the skin

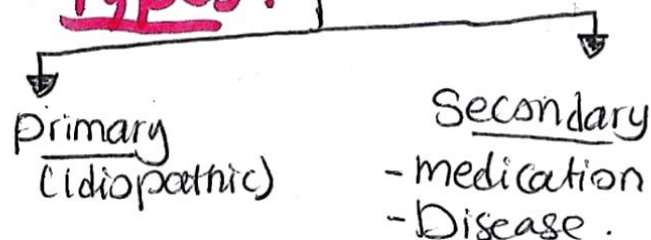
Cytologic inclusions & other morphologic changes of diagnostic importance in the sweat glands

Disorder	Microscopic changes
Hypothyroidism	PAS-positive, diastase-resistant granules in secretory cells.
Lymphoma - Multiple myeloma - Heat stroke	Vacuolar degeneration of secretory cells or ducts.
Mucopolysaccharidoses	Vacuoles in secretory cells.
Sphingolipidoses	Lipid inclusions in secretory cells.
Trisomy 13, 18 or 21	Differences in duct: Secretory coil length.
Severe acute respiratory syndrome (SARS)	Viral particles in lumen of sweat gland tubule.
Uremia	Reduced size of sweat glands.

❖ Hyperhidrosis ❖

• D.f: excessive production of eccrine sweat.

• Types:



• 1ry hyperhidrosis •

• most common Type

• Volar or axillary

• Diagnostic Criteria:

- ↳ Focal, visible, excess sweating
- ↳ present at least for 6 months
- ↳ No apparent 2ry causes
- ↳ At least 2 of the following:-
 1. Bilateral - Symmetrical
 2. Impaired activity of daily life
 3. at least one episode/week
 4. Age of onset < 25yr
 5. +ve family History
 6. Stop During Sleep.

• Axillary hyperhidrosis is D.f. overactivity of eccrine glands
Unlike axillary odor → mainly Apocrine in origin

• palmo plantar Hyperhidrosis :-

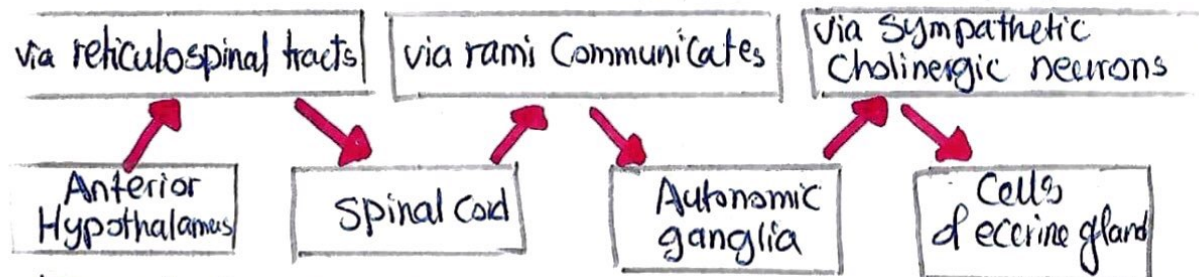
may be inherited in autosomal dominant manner

Volar (palmo plantar) hyperhidrosis (Fig. 1)	Axillary hyperhidrosis
50-60%	30-50%
Onset is during childhood.	Onset is at or soon after puberty.
Entire palm and sole, the lateral aspects, tips and distal dorsal skin of the fingers.	Right axilla usually produces sweat > left.
	Odor (axillary bromhidrosis) is usually absent.

Both types may coexist.

• 2ry Hyperhidrosis •

↳ Localized
↳ Generalized
- Neural impulses for sweating :- (Sudomotor impulses)



- Divided into categories:- Based on the source of the neural impulse driving the response

↳ Cortical
↳ Hypothalamic

↳ medullary
↳ Spinal cord

↳ Local
↳ non-neural in origin

1 Cortical

- Isolated sweating of palm-soles
- Due to: Cortical excitation By:
 - ↳ emotional
 - ↳ sensory stimuli
- Several disorders of Cornification and other genodermatoses affecting palmar/plantar skin are associated with Volar hyperhidrosis:
 - ↳ PPK
 - ↳ epidermolysis bullosa simplex
- ptise Hereditary autonomic Neuropathies → Can also exhibit Cortical Hyperhidrosis

2 Hypothalamic (thermal)

- The hypothalamus Responsible for → Thermoregulation.
- elevation temperature of Body Blood By OBF or more
↓
excites the hypothalamic nuclei
↓
vascular Dilation + generalized sweating

- it may also occur Due to: Direct hypothalamic Stimuli e.g

- ↳ pheochromocytoma
- ↳ medications
- ↳ Toxins

- Maybe → Nocturnal OR → Diurnal

3 Medullary (gustatory)

Taste Receptors are the source of the afferent impulses that stimulate sweating

physiologic

- Triggered By:
 - ↳ Spicy Foods
 - ↳ alcohol
 - ↳ Citrus fruits
- Mediated Through: Reflex arc impulses from taste bud Receptors travel via glossopharyngeal nerve to nuclei within the medulla oblongata
↓
leads to provocation of sweating + Erythema on: upper lip
Cheeks
Rather than the usual Response of Salivation
- Familial tendency
- No other Concurrent local or systemic signs or symptoms

pathologic

- occur when disrupted nerves for sweat → connect e nerves for Salivation.
- Caused By:
 - ↳ injury or disease of parotid gland (Frey's Syndrome)
 - ↳ Trauma to Sympathetic Trunk
 - ↳ CNS disorders:
 - encephalitis
 - Syringomyelia

4 Spinal Sweating :- (Cord transection)

- Spinal disorders Result in:

- Lack of thermal Sweating Below the injury

- Unusual patterns of Hyperhidrosis

- Bouts of "mass reflex Sweating" →

↳ initiated in the skin Below the level of the Cord interruption.

↳ most intense in skin segments Close to the level of the Transection

↳ Mass reflex sweating → doesn't occur in transection below T8 - T10.

5 Autonomic Dysreflexia :-

↳ Direct Stimulation
↳ e.g. electrical - physical
↳ Drug induced.

→ Sympathetic axon → Cause Sweating
→ may be seen with inflammatory skin disease

6 Compensatory :-

- Anhidrosis in one area → Cause hyperhidrosis in another
- Miliaria - Diabetic neuropathy } well-known Causes
- Sympathectomy

7 Non - neural :-

1 - Direct stimulation of eccrine glands → Cause sweating

2 - Triggers include: → Local heat
→ Drugs :- Acetylcholine - Insulin
→ Vascular Tumors

Causes of generalized hyperhidrosis	Causes of localized hyperhidrosis
<ul style="list-style-type: none"> • Febrile infective illnesses: TB, malaria, brucellosis, etc. • Metabolic diseases: DM, hyperthyroidism, hyperpituitarism, hypoglycaemia, pheochromocytoma. • Menopause. • Underlying solid malignancy and lymphoma. • Parkinson's disease. • Congestive heart failure. • Neurological disorders. • Peripheral neuropathies, brain diseases, ... • Episodic hypothermia with hyperhidrosis. • Generalized hyperhidrosis without hypothermia. • Drugs: e.g. Fluoxetine. 	<ul style="list-style-type: none"> • Spinal cord injury. • Intrathoracic neoplasia. • Gustatory hyperhidrosis. • Functional and true sweat gland nevi. • Sweating associated with local skin disorders: <ul style="list-style-type: none"> ◦ Glomangioma. ◦ Blue rubber bleb nevi. ◦ Pachydermoperiostosis. ◦ Pretibial myxoedema. ◦ POEMS syndrome. ◦ Burning feet syndrome. • Compensatory. • Idiopathic unilateral circumscribed hyperhidrosis.

Drugs that Can Stimulate eccrine Sweating :-

- 1] Direct Acting cholinomimetic Agents
Acetyl choline
- 2] Cholinesterase inhibitor
Anti-Alzheimer's drugs
Anti-myasthenic
- 3] Adrenomimetic Agents
- Dopamine
- epinephrine
- Norepinephrine
- 4] Anti-diabetic hypoglycemic :-
- Insulin - Sulfonylurea
- 5] CNS Stimulants:
Amphetamines - Caffeine
- 6] Antidepressants:
MAOIs - SSRIs - tricyclics
- 7] Anti-psychotics:
- 8] Antipyretics: NSAIDs
- 9] Opioids

- Diagnosis:

1] Differentiate Between primary and Secondary

History
Physical exam
lab testing

Laboratory test	Disease
Serum electrolytes, BUN, creatinine	Renal disease (rare)
Blood glucose level	Diabetes mellitus
Thyroid function tests	Hyperthyroidism
Skin test for tuberculosis (e.g. PPD)	Tuberculosis
Chest X-ray	Tuberculosis, neoplasm
Complete blood count	Infection
Sedimentation rate	Infection, neoplasm, inflammatory disease
Antinuclear antibodies	Autoimmune connective tissue disease
Urinary catecholamines	Pheochromocytoma

2] The severity of the condition

	Palmo-plantar hyperhidrosis	Axillary hyperhidrosis
Mild	Moist palm or sole without visible sweat droplets.	Sweat stains on clothing: 5-10 cm.
Moderate	Sweating toward the fingertips.	Sweat stains on clothing: 10-20 cm.
Severe	Drip sweat.	Sweat stains on clothing: >20 cm.

3] Colorimetric techniques as:- Starch iodine :- Iodine solution e.g (2.5% in alcohol) → applied to clean shaved skin → Dry → then starch powder (cornstarch) → The mixture turn Blue/Black in site of sweating

4] To document amount of sweat produced → Gravimetric technique → Evaporative measurement → Infrared thermography

Treatment of hyperhidrosis

	Therapy	Frequency & recommended dose	Side effects	Duration	Comments
First-line	Topicals <ul style="list-style-type: none"> Aluminium chloride hexahydrate – 6.25% (e.g. Xerac AC®), 15% (with 2% SA), 20% (e.g. Drysol®). Aluminium chloride – 12% (e.g. Certain Dri®), Zirconium salts. Aldehydes. 	Use nightly for 3-5 nights, then every few days as needed.	<ul style="list-style-type: none"> Burning. Irritant contact dermatitis. 	Days	<ul style="list-style-type: none"> Blocks sweat ducts. Aldehydes not recommended due to sensitization. Zirconium salts may be effective in axillae, but not volar surfaces.
Sec-ond-line	Iontophoresis	2-3 times a week.	Discomfort during procedure.	Days	Blocks sweat ducts.
	Botulinum toxin A	Every 4-6 ms.	<ul style="list-style-type: none"> Discomfort during injection Weakness of underlying muscles. 	Months	Prevents release of acetylcholine.
	Oral therapy <ul style="list-style-type: none"> Oxybutynin Glycopyrrolate 	As needed	<ul style="list-style-type: none"> Dry mouth & urinary retention most common; also confusion & decreased mental status. 	Hours	<ul style="list-style-type: none"> Anticholinergic Anticholinergic
	Clonidine	• 0.1-0.3 mg bid	<ul style="list-style-type: none"> Hypotension, rebound hypertension. 		<ul style="list-style-type: none"> α2-adrenergic agonist.
	Propranolol	• 10-40 mg bid	<ul style="list-style-type: none"> Hypotension, bradycardia, hyperhidrosis with long-term use. 		<ul style="list-style-type: none"> β-adrenergic blocker.
	Clonazepam	• 0.25-0.5 mg bid	<ul style="list-style-type: none"> Sedation. 		<ul style="list-style-type: none"> Anxiolytic.
Third-line	Local excision	Once	Scarring	Permanent	Last resort
	Sympathectomy		<ul style="list-style-type: none"> Compensatory hyperhidrosis. Horner's syndrome. 	Usually permanent	

bid, twice daily; SA, salicylic acid.

✚ Botulinum Toxin type A :- (BTA)

- For the treatment of axillary hyperhidrosis :-

→ Mechanism of Action :

BTA prevent release of acetylcholine from Cholinergic neurons

→ Effect :

Injection into Hyperhydrotic skin will produce
→ near anhidrosis for 4-6 months

→ Side Effects : (Short lived)

↳ Muscle weakness → of intrinsic muscles of the hands or feet → Resolve over 2-5 weeks

↳ Compensatory Hyperhidrosis Not observed

→ Both Botox and Dysport have similar perceived reduction of sweating in the treatment of primary axillary Hyperhidrosis
Using : Conversion factor of 1:3

100 units Botox vs 300 units Dysport)
and primary Palmar hyperhidrosis using
Conversion Factor 1:4

→ It of axillary hyperhidrosis with B. toxin A reconstituted in lidocaine OR Normal [20]

Saline → was described in a randomized Side-By-Side - double Blind study.

↓
The Result were the same
- associated @ significant reduced pain.

- Laser treatment of Hyperhidrosis

- Photo thermal destruction of sweat glands (desquamation and Rupture of sweat gland)

- Using : → Subdermal Nd:YAG laser
Safe and effective

- Sweat Reduction is proved to be stable over period of upto 24 months.

- treatment of axillary hair using 1064 Nd:YAG laser → Cause hyperhidrosis and That effect is Not Transient

- Microwave thermolysis

- Microwave technology → well suited for targeting sweat glands.

- allowing protection for Both : upper skin layer and structures Beneath the S.C fat.

• D.F: absence of sweat from the surface of skin in the presence of appropriate stimuli

• Causes:

Neuropathic

- Hysteria
- Disease of tumor:-
 - Hypothalamus
 - spinal cord
 - peripheral nerves
- e.g: Leprosy, DM, alcoholism, Sympathectomy

Sweat gland Disturbance

- Aplasia
- Congenital Ectodermal defect
- Ichthyosis
- Atrophy:-
 - Scleroderma
 - Acrodermatitis atrophicans
- plugging of sweat Duct:-
 - miliaria
 - lichen planus
 - Psoriasis

Idiopathic

- Neonatal
- Sweat gland fatigue
- Idiopathic acquired anhidrosis

• Sweat as predisposing influence on the Skin Disease:

- 1- wet skin → more permeable than Dry skin
- 2- wet skin → more likely to develop Contact allergies than Dry skin

3- Anhidrosis

3- Several diseases are aggravated By:

Sweat:

- Hailey-Hailey Disease
- Grover's disease
- Folliculitis
- Pitted Keratolysis

3- Granulosis Rubra Nasi

- Rare autosomal Dominant
- Onset: During the first decade of life
- Resolve: at puberty
- Clinically:
Hyperhidrosis of nose, followed by Macular Erythema, Papules - Vesicles
- may extend to: Cheeks, lips - upper chin
- treatment:
Drying lotions - Reassurance

❖ Diseases of Apocrine Sweat glands ❖

- Fox-Fordyce disease: [Apocrine Miliaria]

- Occur mainly in women soon after puberty
- Ch·ch By: → the presence of Itchy - discrete firm, follicular papules dome shape - skin colored in area which Apocrine glands are found
 - Axillae
 - areola
 - anogenital Region

- The condition improve in:
During pregnancy → D/t ↓ apocrine gland secretion.

• Pathogenesis + Histopathology :-

- Hormonal disturbances → formation of a "Keratotic plug" in the distal Apocrine duct
- Followed By Rupture of the Apocrine Duct.
- Local microvesicle formation
- paracutaneous Acanthosis follow.
- inflammatory infiltrate → at dermis

• Treatment :

1. Topical, intralesional Corticosteroid → 1st line
2. Topical tretinoin
3. Topical clindamycin (twice daily)
4. Isotretinoin
15-30 mg/day → 4 months
- Short Term success
- Condition Returns 3 months after stop
5. Hormonal therapy in women
6. UViolet light
7. dermabrasion
8. Surgical excision → non Curative

Acne

	Acne vulgaris	Lupus miliaris disseminates faciei
CP	<ul style="list-style-type: none"> - Comedones, papules, pustules, nodules, cysts - Face, chest, shoulders, upper back - Post-inflammatory erythema & pigmentation - May heal with scarring - Seborrhea 	<ul style="list-style-type: none"> - Discrete, reddish papules - Face mainly eyelid, cheeks, upper lip - Absent erythema & telangiectasia - Involuting spontaneously with pitted small scars
HP	<ol style="list-style-type: none"> 1- Comedone → keratinous debris, micro-organism, hair, sebum 2- papules → lymphocytic perifollicular infiltrate 3- Rupture of follicle wall → escape of contents → aggregation of neutrophils → pustules & nodules 	<ol style="list-style-type: none"> 1- Large tubercle → epithelioid cells & giant cell 2- Central caseation necrosis 3- Peripheral inflammatory infiltrate
Pathogenesis	<ol style="list-style-type: none"> 1- Increased sebum production 2- Ductal hypercornification 3- Proliferation of P.acne 4- Inflammation 	<ol style="list-style-type: none"> 1- No evidence support tubercle etiology 2- It may be related to rasacea
ttt	Anti-acne agents	Tetracycline, minocycline, isotretinoin

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Acne

	Neonatal acne	Infantile acne
<ul style="list-style-type: none"> - May be present at birth or develop during the 1st few months - More common in males - Mild & regress spontaneously by the age of 6 months - Not associated with significant scarring or increased incidence of acne later in life 	<ul style="list-style-type: none"> - Begins between 3rd & 6th month - More common in males - Severe nodules & cysts & may persist to age of 5 years - Associated with significant scarring or increased incidence of acne later in life - Associated with vililizing tumors 	<ul style="list-style-type: none"> - Intrinsic hormonal imbalance ↑ testosterone, LH, DHEA
Pathogenesis:	<ul style="list-style-type: none"> - Species of malassezia - Response to topical ketoconazole - Sebum excretion: high level 	

Amylee

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Acne vulgaris	Acneiform eruption
<ul style="list-style-type: none"> - Comedone, papule, pustule, nodules, cyst - Face, chest, shoulders, upper back - Age: male: 16-19, females: 14-16 - Chronic inflammatory disorder - Acne variants: - ttt: anti-acne agent 	<ul style="list-style-type: none"> - Papule, pustule - Not confined to the usual sites - Passing acne age - Sudden onset - Types: ... - Resolve slowly with withdrawal of the cause

(12)

QAcne:

- 1- Pathogenesis of acne vulgaris.
- 2- Give an account on acne vulgaris
- 3- Give an account on Acne variants.
- 4- Pathogenesis, clinical types & therapeutic modalities in acne vulgaris.
- 5- Compare between acne conglobata & acne fulminans.
- 6- Compare between Acneiform eruptions & acne vulgaris
- 7- Compare between neonatal acne & infantile acne
- 8- Management and Medical ttt of nodulocystic acne.
- 9- Acneiform eruptions.

- 10- Preoperative considerations of laser ttt of acne scar
- 11- Compare: AV & drug induced acne
- 12- Compare: AV & lupus miliaris disseminated faciei
- 13- CP & management of rosacea
- 14- Eye manifestations of rosacea
- 15- Eccrine & apocrine sweat gland: distribution, histological shape, location, mechanism of secretion, diseases affecting them
- 16- Hyperhidrosis, causes, clinical variants & ttt



Acne

Acne

acne

acne varint ?

1. acne fulminant clinically? Itt?

2. classification of acne ?

inflammatory and non inflamtory

a. mild,mod.severe

Gram negative folliculitis

Isotretinoin cumulative dose

مهم جدا امتي ندى اقل من 5. مجم في اليوم في الـروز سيشيا

Acne fulminans and conglobata ?

Why is hemorrhagic nodule in acne fulminans ? vascular

Systemic manifest of acne fulminans ?

Lupus miliaris ?

Compare Lupus miliaris and acne ?

Pathogenesis of acne ?

syndrome associated with acne?

Emergency of acne ?

Type of comedo of acne conglobata ?

Reason and ttt of an fulminans and conglobata?

Low dose uses of isotretinoin?

اكتب رويشة لحب الشباب ؟ ده سوال رويش اف الشفوي

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